Advances in Spinal Cord Stimulation

SPINAL CORD STIMULATION is a technique in which electrical stimulation is applied posteriorly to the spinal cord. It was first employed 30 years ago to manage chronic pain limited to several contiguous spinal segments. Although spinal cord stimulation is nondestructive and reversible, because of technical limitations, it did not gain wide acceptance until this decade. Recent improvements in equipment and in patient selection have decreased the risk of complications and improved efficacy.

Percutaneous placement now permits lead positioning without general anesthesia. Patients then provide important feedback during lead placement, enhancing the ability to locate the precise area of the cord responsive to stimulation. Percutaneous lead placement also permits a trial stimulation without committing the patient to incision for the introduction or removal of equipment.

Using pacemaker technology, internal pulse generators were developed for a totally implanted, self-contained system programmed externally. Alternatively, internal pulse receivers are implanted and powered by radiofrequency through a 7.6-cm (3-in) antenna above the skin, a system that does not require surgical intervention when the battery fails. Multilead systems allow the delivery of complex stimulation patterns for patients who are unresponsive to stimulation by a single lead. Flat, wide leads for more varied stimulation arrays and wider areas of coverage can be placed by laminectomy.

Patient selection is an essential component of managing pain syndromes with spinal cord stimulation. Patient suitability is assessed by psychometric testing, such as a specially adapted, validated version of the Minnesota Multiphasic Personality Inventory. A multidisciplinary patient evaluation and refined trial techniques allow a better prediction of which patients are likely to benefit from the procedure.

Spinal cord stimulation is also an effective adjuvant to standard therapies for refractory angina pectoris. Epidural leads are placed near T-1, and stimulation produces paresthesias in the aching area. Spinal cord stimulation substantially improves exercise capacity and quality of life while reducing the number of anginal attacks, ischemic electrocardiographic signs, and nitrate consumption. There is no evidence that it conceals the signs of acute myocardial infarction. In Europe, this technique is a routine supplement to conventional medical and surgical therapies for angina.

Similarly, spinal cord stimulation produces an antiischemic effect in peripheral arterial and severe vasospastic disease of the limb, including that of patients refractory to standard medical and surgical therapies. Patients with residual vascular compliance are most likely to respond to the procedure. In patients with peripheral vascular disease, spinal cord stimulation may increase exercise tolerance, aid in the healing of ischemic ulcers, and increase microvascular flow. Despite this, the primary target of spinal cord stimulation therapy in the United States in patients with peripheral vascular disease remains the management of pain associated with the disease.

In summary, pain from ischemic vascular diseases and chronic pain from syndromes such as the failed back, phantom limb, and complex regional pain syndrome I (reflex sympathetic dystrophy) can be effectively managed with spinal cord stimulation. To date, there have been about 100,000 implantations of spinal cord stimulation. It is likely that the use of the procedure will increase in the future because of improved efficacy and the increased number of applications.

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Use of β-Blockade to Prevent Death After Noncardiac Surgery

IN THE UNITED STATES each year about 30 million patients have noncardiac operations. Of these, about 1 million have diagnosed coronary artery disease, 2 to 3 million have two or more major risk factors for coronary artery disease, and another 4 million are older than 65. Despite advances in the diagnosis and treatment of coronary artery disease, the perioperative morbidity and mortality in this group remain high. The incidence of intraoperative ischemia is between 20% and 63%, and that of postoperative infarction can be as high as 37% with an associated mortality of 40% or higher. Of all possible predictors of an adverse outcome, postoperative ischemia has been identified as the most important, conferring a ninefold increase in the odds of having cardiac death, myocardial infarction, or unstable angina and a twofold risk of long-term sequelae. Thus, efforts at reducing adverse cardiac outcomes have concentrated on the preoperative evaluation and on reducing the incidence of postoperative ischemia.

In a number of studies, the effects of techniques for reducing perioperative myocardial ischemia, a possibly reversible cardiac risk factor, have been examined: anesthetics, postoperative sedation, prophylactic nitrates, calcium channel blockers, and β-blockers. Of these, intensive postoperative sedation, β-blockade, α_2-agonists, and adenosine analogues have shown reductions in the incidence or severity of perioperative myocardial ischemia. Until now, however, none of the clinically available therapies have shown a difference in mortality.

Recently a 200-patient, randomized, placebo-controlled, clinical trial showed that the prophylactic perioperative administration of atenolol reduced mortality after discharge from the hospital. The major reduction in the number of deaths from cardiac causes occurred during the first six to eight months (0% versus 8%, $P < .001$).